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ON THE SCIENTIFIC ADMINISTRATION OF ANÆSTHETICS.

By S. Thompson Rowling, M.D.

Lecturer in Anæsthetics, University of Leeds; Senior Anæsthetist, General Infirmary at Leeds.

DURING the present year research work has been carried on with the object of placing the administration of anæsthetics on a definite scientific basis.

To this end, an improved inhaler has been made, and the actual percentages of ether, carbon dioxide and oxygen which are inhaled at various positions of the ether valve and air slot have been worked out in a physiological laboratory by Haldane's gas analysis apparatus.

The results are engraved on the inhaler.

By this research, the percentages of the vapours of ether, carbon dioxide and oxygen in the anæsthetic atmosphere may be read off or calculated throughout the administration.

An anæsthetic table has also been arranged to supply the gases, oxygen, carbon dioxide and nitrous oxide from cylinders clamped to the table.

Three dry flow-meters are used to indicate the rates of flow of the three gases measured in litres per minute.

A simple oil-chloroform percentage vaporiser, the use of which is optional, completes the equipment.

The Chief Volatile Anæsthetics and their Pharmacology.

The chief volatile anæsthetics used in England are nitrous oxide, ethyl chloride, ether and chloroform. Clinical experience teaches that nitrous oxide gives excellent results for cases

not requiring muscular relaxation, such as breast cases, but that it cannot be relied upon to obtain muscular relaxation and is therefore not suitable for abdominal work. Ethyl chloride is rapid and safe for short administrations, but is not suitable for prolonged anæsthesia. Ether is the best anæsthetic for all cases requiring muscular relaxation, but hitherto has been difficult to administer on account of certain disadvantages. Chloroform has a limited sphere as the handmaid of ether. It should be used sparingly, in definite doses, as an adjuvant or corrective in the anæsthetic prescription.

Dosage.

The relative pharmacological strengths of the chief volatile anæsthetics used in England may be stated as:

Chloroform		Ether		Ethyl Chloride		Nitrous Oxide.
1%	=	7%	=	? 19.5%	=	85%

To *obtain* anæsthesia an average dose of 3.5 per cent chloroform or 24 per cent ether may be needed.

To *maintain* anæsthesia an average dose of one per cent to two per cent chloroform or seven per cent to ten per cent ether, is needed.

Two other ingredients of the anæsthetic atmosphere are essential for a completely successful administration: these are oxygen and carbon dioxide.

The use of Oxygen during the administration of Anæsthetics.

Research work by J. S. Haldane, and others, demonstrates the fact that even slight anoxæmia will cause damage, chiefly to the nervous and circulatory systems. Barcroft states that lack of oxygen produces marked effects in the higher centres of the brain in less than fifteen seconds.

The causes of anoxæmia during anæsthesia are:—

1. Shallow breathing.
2. The recumbent position.
3. Paralysis of the nervous control of the bronchial muscle.
4. Paralysis of the respiratory centre.

5. Obstruction in the respiratory passages.
6. Reduction of the oxygen content in the anæsthetic atmosphere.
7. Initial hypernœa—in open mask administrations.

Clinical research by means of Haldane's gas analysis apparatus shows that during an actual administration, the oxygen content of the anæsthetic atmosphere is always reduced. The oxygen percentage in air is 20.96 per cent., but in the anæsthetic atmosphere of an ether inhaler without the bag, it is reduced to 17.5 per cent when the ether valve is at full, and with the bag it may be reduced to less than 10 per cent. In the anæsthetic atmosphere beneath an anæsthetic mask covered by gauze or tetra-cloth—the so-called open method—the oxygen percentage is always reduced: one sample gave 17.54 per cent and others to as low as 13.45 per cent.

This research shows that in all administrations of anæsthetics there is some anoxæmia unless oxygen is added to the anæsthetic atmosphere. The effect of anoxæmia on the nervous and circulatory systems, on which life depends, is one of profound depression.

To eliminate anoxæmia throughout the administration, oxygen must be added to the anæsthetic atmosphere. The usual rate at which oxygen should be given is one litre per minute. Taking the normal respiratory rate at six litres per minute, it will be seen that the proportion of added oxygen is 16.5 per cent.

The actual rate of flow is read off by means of a dry flowmeter, and is adjusted to the patient's needs according to the scale on the inhaler.

The use of Carbon Dioxide during the administration of Anæsthetics.

The great importance of carbon dioxide in the blood has only recently been recognized. The bag of a Clover's inhaler is regarded by many anæsthetists only as a means of concentrating the ether vapour given off from the inhaler, and of preventing its escape. As a means of increasing the carbon dioxide in the blood, its use is not generally recognized, and

yet this use is of the utmost importance, and it is essential that it should be understood and appreciated.

Henderson has investigated this subject very carefully and in a series of articles on "Acapnia and Shock" published in *the American Journal of Physiology*, summarizes his results. Carbon dioxide must not be regarded merely as a waste product of tissue metabolism: it acts as a regulator on various functions—on the pulse rate and circulatory tone, on intestinal peristalsis and on the mental functions. A slight reduction of carbon dioxide in the blood causes a quickened pulse rate; a further reduction, tachycardia; and an extreme reduction, cardiac failure.

A slight reduction of carbon dioxide, maintained for some time, causes a condition apparently identical with surgical shock—rapid feeble pulse, lowered blood pressure, shallow breathing, and cerebral depression.

In a lecture on "Respiration in Anæsthesia: "Control by Carbon Dioxide" (*Brit. Med. Journ.*, December 19, 1925) by the same author, the subject is exhaustively dealt with. The summary and conclusions of this lecture are that "The essentials of good anæsthesia are rapid induction, uniform concentration of vapour, and rapid termination. In the drop method of administration the vaporisation of a liquid anæsthetic is often irregular. A more reliable and more uniform method of producing the vapour in proper concentration in air is desirable and is afforded by many of the forms of apparatus introduced recently.

The use of a controlable amount of re-breathing is also advantageous in the maintenance of a nearly uniform concentration of the anæsthetic vapour and of oxygen and carbon dioxide.

An almost complete control of respiration is afforded the anæsthetist by the use of carbon dioxide mixed with oxygen. By its use rapid induction and termination of anæsthesia and any desired volume of breathing and depth of anæsthesia are at his command.

It is not the amount of anæsthetic in the body which determines anæsthesia, but that in the nervous system. The amount in the nervous systems at all times follows closely the concentration in the arterial blood; and the concentration

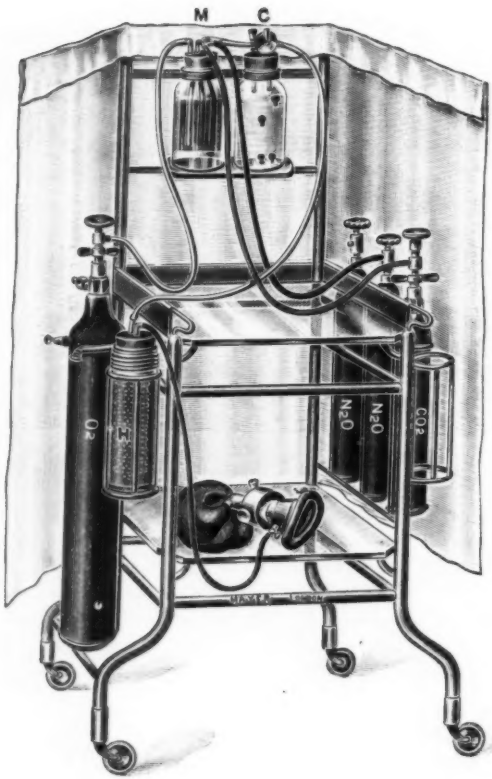
in the arterial blood is in turn determined by the volume of the breathing and the concentration of the anæsthetic vapour in the air inhaled. Thus control of respiration is the essential element in the control of anæsthesia.

The respiration of an approximately normal content of carbon dioxide in the lung air and in the blood and tissues of the body at the termination of anæsthesia, probably tends strongly to prevent the development of a post-anæsthetic pneumonia. It is also the means of restoring a nearly normal acid base balance in the blood."

Experimental work by Levi, "Clinical Use of Carbon Dioxide with Oxygen (*Journ. Amer. Med. Assoc.*) has shown that in animals, when the circulatory bulbar centres were temporarily paralysed by chloroform or ether or operative trauma, or by a combination of these causes, the inhalation of carbon dioxide and oxygen brought about an immediate recovery. In hundreds of cases the almost invariable result was a rapid recovery. Clinical observation and experience is completely in accord with these conclusions: their recognition and practical application in the operating theatre constitute one of the fundamental factors of a rapid, safe and profound narcosis.

One example will suffice. A. B. Male, aged 55, Carcinoma Ventriculi. Blood transfusion November 6, 1928. Operation November 8, 1928. Gastrectomy. Time, two hours ten minutes. Patient collapsed on table at end of operation, apparently dying, but restored by CO_2 20 per cent + oxygen 80 per cent, with hot cloths over precordium.

Experimental evidence by Hill and Fleck, "The Effect of Excess of Carbon Dioxide or want of Oxygen on the Respiration and the Circulation" (*Amer. Journ. of Physiol.*) proves that carbon dioxide is a stimulant of respiration up to 35 per cent, beyond which respiration is depressed; also that circulation is stimulated up to 22 per cent, beyond which it is depressed. Research to ascertain the percentage of carbon dioxide in re-breathed air from the bag of an inhaler after one half-hour re-breathing, with oxygen added to prevent anoxæmia, gave 22.12 per cent as the result of analysis by Haldane's gas apparatus. By the use of a specially devised slotted angle-piece, any percentage up to this amount can


M
C
O2
N2O
N2O
CO2

stimulating of the nervous, circulatory and respiratory systems which an excess of carbon dioxide brings about.

The use of oxygen and carbon dioxide together prevents both anoxæmia and apnœa and eliminates the serious depression of the nervous, circulatory, and respiratory systems, which both anoxæmia and apeœa produce.

This doubly beneficial effect, with the doubly harmful effect eliminated, is obtained by giving oxygen continuously throughout the administration, together with a regulated re-breathing or the actual addition of carbon dioxide, to increase the carbon dioxide percentage. Oxygen prevents the cyanosis of re-breathing—carbon dioxide prevents the apnœa of oxygen.

In the section on Apparatus and Technique will be found a description of how this is carried out clinically with the greatest ease and percentage precision.

To maintain the circulatory and respiratory systems, carbon dioxide and oxygen are essential: the percentage of carbon dioxide being from one to 20 per cent, and of oxygen from 21 to 80 per cent, according to circumstances.

The Anæsthetic Table.

The Table is designed to supply an atmosphere of oxygen, nitrous oxide or carbon dioxide, or of these gases in any percentage combination, and with or without chloroform vapour of definite strength. The gases oxygen, nitrous oxide and carbon dioxide are obtained from cylinders clamped around the table. Three dry flow-meters, graduated in litres per minute, enable the anæsthetist to administer these gases either alone or in any combination and at any desired rate of flow.

The chloroform vapour of definite strength is obtained by vaporization from a bottle containing chloroform and an inert neutral, non-volatile diluent: liquid paraffin is the diluent chosen. Undiluted chloroform, under similar conditions, gives off 18 per cent of vapour, but when diluted in definite proportions, a definite and much smaller percentage is vapourized.

The data are as follows: The specific gravity of chloroform is 1.497. The specific gravity of liquid paraffin is .88; a mixture of the two liquids has an intermediate specific gravity. A mixture of chloroform 6 drs. with paraffin liquid

12 ounces has a specific gravity .914, temperature 70 degrees F., and vapourizes 3.5 per cent chloroform. A specific gravity bead .914 (Red) immersed in the mixture, rises when the proportion of chloroform exceeds this amount and falls when it is less—in other words, when the Red bead rises the percentage of chloroform vaporized exceeds 3.5 per cent: when it falls, the percentage is less than 3.5 per cent.

Similarly, a mixture containing only 5 drs. of chloroform to 12 ounces liquid paraffin has a specific gravity of .908 and vaporizes three per cent of chloroform—a specific gravity bead .908 (Blue) rises when the proportion of chloroform exceeds this amount and falls when it is less—in other words, when the Blue bead rises the percentage of chloroform vaporized exceeds three per cent: when it falls, the percentage is less than three per cent.

A mixture containing 4 drs. of chloroform has a specific gravity .903 and vaporizes 2.5 per cent of chloroform—a specific gravity bead .903 (Yellow) rises when the percentage of chloroform vaporized exceeds 2.5 per cent and falls when the percentage is less than 2.5 per cent.

A mixture containing 3 drs. of chloroform has a specific gravity .897 and vaporizes two per cent of chloroform—a specific gravity bead .897 (Green) rises when the percentage of chloroform vaporized exceeds two per cent and falls when the percentage is less than two per cent.

Thus, when all the specific gravity beads are floating the percentage of chloroform being vaporized exceeds 3.5 per cent, when all the beads sink the percentage is less than two per cent. When one sinks and three float, the percentage is between 3 per cent and 3.5 per cent: when two sink and two float, the percentage is between 2.5 per cent and 3 per cent, and when three sink and one floats, the percentage is between 2 per cent and 2.5 per cent. The position of the beads shows at a glance the percentage of chloroform which is being vaporized. These percentages have been worked out by Waller's Densimetry method.

This bottle method is a most simple means of obtaining clinically a definite percentage of chloroform vapour on the "Plenum" system—much simpler than the methods devised by Dubois, Waller, Collingwood or Alcock. It is accurate

for all practical purposes and fool-proof in that it is impossible to give a higher percentage of chloroform vapour than that indicated by the specific gravity beads. The rate of flow of the vaporizing gas or gases determines the actual percentage which the patient is inhaling: taking the average volume of respiration at six litres per minute, a rate of one litre per minute would give one-sixth of the percentage vaporized.

Any decomposition of chloroform in the oil-chloroform mixture, due to oxidation by light or oxygen bubbling would produce phosgene gas. A sample oil-chloroform mixture after some weeks' use in the operating theatre has been analysed and found to contain minute traces of this gas. C. K. Ingold, D.Sc, F.R.S., and H. Burton, M.Sc., Ph.D. who have undertaken this part of the research report as follows:

"The addition of a small amount . . . of α-naphthylamine would absorb phosgene." A later report on an oil-chloroform mixture with α-naphthylamine added states that "the turbidity present in the mixture of chloroform, liquid paraffin and α-naphthylamine is α-naphthylamine hydrochloride, this demonstrating that a small amount of phosgene has been produced during your work with it." "The re-action between phosgene (carbonyl chloride) and α-naphthylamine hydro- proceeds according to the equation $2C_{10}H_7NH_2 + COCL_2 = 2HCL + (C_{10}H_7NH)_2CO$. The hydrochloride liberated reacts with any excess of α-naphthylamine forming α-naphthylamine hydrochloride, i.e. $C_{10}H_7NH_2HCL$, whilst the *s*-di α-naphthylamine carbanide will have little or no volatility.

The actual investigation was as follows:— "When the turbid liquid paraffin solution was mixed with an equal volume of ether the turbidity remained unaltered, thus indicating that the substance causing it is insoluble in ether. When the ethereal solution was shaken with 20 ccs. water a yellow aqueous extract was obtained, and the ethereal extract became clear. This indicates that the substance causing the turbidity is soluble in water. Part of the aqueous extract was treated first with nitrous acid and then with an alkaline solution of β-naphthol. A red dye was produced. This shows definitely that the aqueous extract contains a water-soluble α-naphthylamine salt, i. e. the hydrochloride. Definite proof that it was the hydrochloride was obtained thus: a portion of the

aqueous extract was treated with silver nitrate solution. A white precipitate of silver chloride, insoluble in nitric acid, was obtained."

As a result of this research, 10 grains α-naphthylamine have been added to the oil-chloroform mixture, thus eliminating the danger of the impurity phosgene gas in the vaporized chloroform.

The table is compact, convenient and practical; covered in on three sides by a sterilized sheet, it fulfils the aseptic requirement of a modern operating theatre; and used with an inhaler or mask for ether it contains all that is essential, nothing that is superfluous, for the construction of an anæsthetic atmosphere which is both efficient and safe.

Note.

The dry-flow meters have been chosen in preference to a sight-feed water bottle for estimating the percentages of oxygen, nitrous oxide and carbon dioxide, because of greater scientific accuracy and easier clinical reading.

Considerable time has been spent in endeavouring to perfect a sight-feed water bottle. It was found that the different gases emerged from the tube perforations at different rates—in other words that a drill of 70 gauge was needed for the oxygen delivery tube, one of 68 gauge for the nitrous oxide and one of 66 for carbon dioxide in order to deliver approximately the same volume of gas in the same time. It was necessary to place the holes in logarithmic order, proportional to the square root of their distances below the water surface. And clinically, the volumes of gas passing through were far less easy to read off.

The Modified Clover's Wide Bore Inhaler.

In devising this modified inhaler, the old well-established and familiar methods of administration have been retained, with simple modifications. Each modification is complete in itself, and may be used apart from any other, and the inhaler may be used precisely like one of standard pattern, if desired.

(*a*) The dome of the inhaler is modified in shape, so as to give a maximum ether capacity with a minimum of space. Above the usual markings, F. $\frac{3}{4}$, $\frac{1}{2}$, $\frac{1}{4}$, o are engraved the actual

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ether percentages given off in these positions of the ether valve lever. The percentages vary considerably with the position of the air-slot on the angle piece, and are given with the air-slot closed, half and open. All these percentages have been worked out by Haldane's Gas Analysis Apparatus in the Physiological Laboratory of the University of Leeds by H. Whitridge Davies, M.B. B.S., from samples taken during actual administrations. They are as follows:

Air full	6%	4%	2%	1%	0	
,, half	8%	7%	3%	3%	0	error ± 2%
,, closed	13%	11%	6%	5%	0	
	F	¾	½	¼	0	

(*b*) The bore of the air shaft passing through the dome is increased in the ratio of 1 to 1.414 in diameter—this doubles the area of the shaft where it is halved by the valve, and so the area of the airway is kept constant, this presenting twice the area of the ordinary wide-bore model. The slots in the shaft are enlarged to give a corresponding mathematical area. The maximum area of the human trachea is 5.07 sq. cm.: the minimum area of the air shaft in Clover's inhaler is 1.9 sq. cm., in Hewitt's wide-bore inhaler it is 3.75 sq. cm., and in the present model it is 10.625 sq. cm.; this is more than twice the maximum area of the trachea, nearly three times the area of Hewitt's wide-bore inhaler and nearly six times the area of Clover's inhaler. The effect of this is that strain on on the respiratory pump is practically eliminated.

Note.—These mathematical calculations have been kindly done by Demonstrators in the Physics Laboratory of the University of Leeds.

(*c*) The inner valve sleeve is in one piece: it is easily removable from below for lubricating, and needs only one short valve lever.

(*d*) A small window is placed in the dome; this enables the ether level to be seen during the administration and thus a constant and sufficient vaporizing surface is secured.

(*e*) The water tank is dispensed with, thus saving weight; the actual weight of the dome is the same as that of a dome of standard pattern, in spite of the doubled air-way.

(*f*) A funnel-ended filler replaces the ordinary air-tight stopper; this enables the dome to be filled or replenished at any time during the administration, without removing the inhaler from the face. The dome remains air-tight when in use, as the lower end of the filler dips into the contained ether.

(*g*) The angle-piece contains a graduated air slot which may be opened to any extent. When open, there is no re-breathing into the bag; when closed, there is full re-breathing and when partly open there is re-breathing to the extent shown by the graduated scale. Beneath this scale are engraved the actual percentages of carbon dioxide when the air slot is closed, one-quarter, one-half, three-quarters or fully open. These percentages vary with the position of the ether valve on the dome and are given with the ether valve at o, ½ and full. These percentages are as follows:—

	Air	o	¼	½	¾	Full	
Ether	o	>6	6	4	2	1	error ± 1
,,	½	>7	6	4·5	3	1·5	
,,	F	>10	9	7	5	3	

The oxygen percentage without added oxygen is found to be 20 minus the percentage of CO_2. Thus in every position of the ether valve and air slot and throughout the administration, the percentages of ether, carbon dioxide and oxygen in the anæsthetic atmosphere can be read off or calculated, and both the strength of the anæsthetic atmosphere and of its carbon dioxide and oxygen constituents can be regulated.

(*h*) The face piece is of the ordinary pattern, but it has a larger air cushion and a tap. The larger air cushion makes the face-piece fit more comfortably and accurately, and the tap is to admit oxygen, nitrous oxide, carbon dioxide or vaporized chloroform.

(*h*) The inhaler is easily and completely dissembled for boiling.

Many anæsthetists prefer an open mask to any form of closed inhaler.

Research has been made into the actual conditions which obtain under an open mask. The results are as follows:—

With one layer of tetra-cloth 40 minims per minute.
Ether 8.79%
Carbon Dioxide no readable difference.
Oxygen 17.54%

With two layers of tetra-cloth 40 minims per minute.
Ether 13.15%
Carbon Dioxide no readable difference.
Oxygen 13.45%

With two layers of tetra-cloth 30 minims per minute.
Ether 11.4%
Carbon Dioxide no readable difference.
Oxygen 15.23%

With two layers of tetra-cloth, Student administering.
Ether 14.35%
Carbon Dioxide no readable difference.
Oxygen 14.91%

With 12 layers of gauze, Student administering.
Ether 9.94%
Carbon Dioxide .001%
Oxygen 17.6%

These figures show that when an open mask is used, the oxygen percentage in the anæsthetic atmosphere is always reduced, and the carbon dioxide percentage is not maintained; that the ether percentage is variable and not definitely known.

The conclusions are that an open mask may be used, if oxygen and carbon dioxide are added to prevent anoxæmia and apnoea, but that the method is not so precise as that of a percentage inhaler.

Artificial Air-way.

The maintenance of a free air-way during the administration of an anæsthetic is absolutely essential to safety and success.

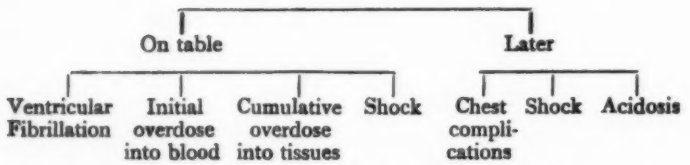
An artificial air-way, modified from Hewitt's has been designed. It consists of an aluminium mouth prop with a short flange which acts as a tongue depressor. When needed, a rubber tube is passed through the air-way as far as is

required to relieve obstruction. In abdominal work, this is changed for a modified Featherstone's air-way as deep anæsthesia is obtained.

Tongue Forceps.

A loop of thread is passed through the tongue by means of the needle blade of the forceps; the double loop is then grasped by the forceps. The instrument hanging on the thread is out of the way of the anæsthetist, but is ready to exert traction on the tongue when required, and does not interfere with the accurate adjustment of the face piece.

DANGERS OF ANÆSTHESIA.



Ventricular Fibrillation is eliminated by Ether plus $CO_2 + O_2$.
Initial Overdose into blood is eliminated by $CO_2 + O_2$ with diminished Ether percentage.
Cumulative Overdose into Tissues eliminated by known and regulated percentage administration.
Chest troubles eliminated by CO_2 Chloroform induction, avoidance of Overdose and rapid recovery.
Shock is eliminated by $CO_2 + O_2$.
Acidosis is eliminated by CO_2.

Comment: The Inhaler and Table meet all these requirements.

The Administration.

A few kindly words give confidence to the patient.

Without delay a warmed and scented Gamgee pad is laid over the face: The pad suggests sleep; it prevents a sight of the inhaler and actual cantact of the face-piece with the skin. The scent blends with the vaporized anæsthetic vapour.

Nitrous oxide or chloroform may be used as the inducing agent.

When chloroform is used, it is vaporized by oxygen and carbon dioxide: the oxygen prevents the sense of suffocation and the carbon dioxide increases the volume of respiration.

The carbon dioxide is turned off when the patient is breathing deeply and the chloroform vaporizer is short-circuited after a few more breaths. During this time ether is poured into the inhaler, and the ether valve gradually opened.

The patient is now inhaling ether, oxygen and carbon dioxide which is continued until full anæsthesia is attained.

The percentages of ether, oxygen and carbon dioxide are then adjusted according to the patient's requirement, to maintain anæsthesia.

A deep anæsthesia, with complete muscular flaccidity but a good pulse and no trace of anoxæmia, may be maintained as long as is required.

When the operation is nearly over, the anæsthetic is rapidly eliminated by giving oxygen and carbon dioxide. The result of this is that the patient has a brisk corneal reflex before leaving the theatre.

The increased breathing not only rapidly eliminates the anæsthetic but ensures thorough lung ventilation.

By giving an excess of carbon dioxide throughout the administration, the acid-base of the blood is maintained, and post-anæsthetic vomiting largely prevented.

To obtain a minimal respiratory excursion during delicate abdominal manipulations, an excess of oxygen and diminution of carbon dioxide, with some deepening of the anæsthesia is required.

The muscular flaccidity obtained is such that preliminary medication is not required, and has not been used for some time. It is found that recovery is delayed, and chest complications are more likely to occur when preliminary medication is used.

The apparatus gives a sense of security, efficiency and scientific precision which is felt by each member of the surgical team, and reacts beneficially on the patient, both directly and indirectly.

ILLUSTRATIVE CASES. (1)

Operation Abdominal pan-hysterectomy.
Surgeon Mr. - - - - -, F.R.C.S.. Place; Nursing home.
Date November 28th, 1928.
Name of patient, Mrs. H - - - -. Age 45. Sex; female.

General condition. Good.
Anæsthetic. Vaporized Chloroform. Ether with added CO_2 and O_2.
Method. Table and inhaler.
Duration 9.8 a.m. to 10.3 a.m.
Quantity Ether six ounces two drams; chloroform half dram.

REMARKS.

9.8 Administration begun.
9.10½ Automatic breathing, E. 13%; CO_2 10%; O_2 25.5%
9.16 Deeply anæsthetized. Pulse 108; resp. rate 42; flaccid. Ether 8%; CO_2 7%; O_2 29.5%
9.22 E. 7%; CO_2 5%; O_2 31.5%. Pulse 98. Resp. 45.
9.29 E. 8%; CO_2 7%; O_2 32.5%
9.45 E. 7.5%; CO_2 6%; O_2 32.5% Correct maintenance dose for this patient.
10.3 Anæsthetic stopped. $CO_2 + O_2$ given.
10.9 Brisk corneal reflex.
10.45 Asleep in bed in Simms' latero-prone position. Pulse 96; Resp. 19.

Comment.

Complete flaccidity, minimal respiratory excursion, no cyanosis.

ILLUSTRATIVE CASES. (2)

Operation. Cholecystectomy, Appendectomy.
Surgeon Mr. ----, F.R.C.S. Place: Hospital. Date; Nov. 29, 1928.
Name of patient Mrs. D. Age 61. Sex; female.
General condition Fair.
Anæsthetic Vaporized chloroform; ether; CO_2 and O_2.
Method. Table and inhaler.
Duration 9.17 to 10.16 a.m.
Quantity. Ether, nine ounces four drams; chloroform 20 min.

REMARKS.

Before anæsthetic, pulse 94; resp. 20.
9.17 Administration begun.
9.19 Automatic respiration—CO_2 stopped.
9.22 Corneal reflex absent. E. 13%

9.22½ Ether 6% (to anticipate initial overdose into blood); O_2 six litres.
9.26 Ether 7%; CO_2 5%; O_2 31.5%.
9.36 Ether 7.5%; CO_2 6%; O_2 30.5%. Pulse 92; resp. 27. Correct maintenance dose for this patient.
9.42 Ether 7½%; CO_2 6%; O_2 30.5%. Pulse 94; resp. 32.
9.48 ,, ,, ,, ,, 92 ,, 36.
9.54 ,, ,, ,, ,, 94 ,, 36.
10.0 ,, ,, ,, ,, 96 ,, 35.
10.15 ,, ,, ,, ,, 106 ,, 43.
10.16 Anæsthetic stopped; O_2 3 litres + CO_2 2 litres given. The effect of CO_2 noted in 15 seconds.
10.18 Corneal reflex present. P.esp. 32, deep and full; Pulse 120.
10.23 Brisk corneal reflex.
10.24 Expiratory phonation.

ILLUSTRATIVE CASES. (3)

Operation Carcinoma sigmoid. Exploratory laparotomy. Colotomy.
Surgeon M. - - - -, F.R.C.S. Place; Hospital. Date; November 29th, 1929.
Name of patient Mrs. S . . . . Age 49. Sex; female.
General condition Spare. Pulse 84; resp. 18; chest clear; blood pressure 155/78.
Anæsthetic Vaporized chloroform; ether; CO_2 and O_2.
Method Table and inhaler.
Duration 10.37 to 11.22 a.m.
Quantity Ether five ounces six drams. Chloroform, not exceeding 20 mins.

Remarks.

10.37 Administration begun. O_2 1 litre + CO_2 1 litre; vaporizing chloroform.
10.38 Ether put into inhaler.
10.38½ CO_2 stopped.
10.39 Chloroform stopped—Automatic breathing. E. 11%; CO_2 10%.
10.40 E. 13%; CO_2 10%; O_2 25%; blood pressure 165/90.
10.42 E. 6% (reduced to anticipate initial overdose into blood)

10.45 E. 7.5%; CO_2 6%; O_2 30.5. Completely flaccid with minimal respiratory excursion.
10.55 Blood pressure 150/72.
11.0 E. 6%; CO_2 6%; O_2 33.5%. Pulse 88; resp. 42. Correct maintenance dose.
11.5 Blood pressure 138/95.
11.15 E. 6%; CO_2 6%; O_2 33.5%. Pulse 82; resp. 46. Blood pressure 135/108.
11.22 Anæsthetic stopped. O_2 3 litres + CO_2 2 litres given. Effect of CO_2 noticeable in 15 seconds.
11.25 Blood pressure 140/110.
11.27 Corneal reflex brisk.
11.29 Phonation.

ILLUSTRATIVE CASES. (4)

Operation For suppurative periostitis of femur.
Surgeon Mr. - - - - -, F.R.C.S. Place; Hospital. Date; November 29, 1928.
Name of patient Miss A. Age 30. Sex; female.
General condition Good. Pulse 78; resp. 12. Blood pressure 120/85.
Anæsthetic Vaporized chloroform; ether; CO_2 and O_2.
Method Table and inhaler.
Duration 11.50 a.m. to 12.15 p.m.
Quantity Ether three ounces four drams. Chloroform—not exceeding 20 mins.

Remarks.

11.50 Administration begun. O_2 1 litre; CO_2 3 litres. Rapid breathing after 14 breaths. Ether added.
11.52 Chloroform stopped.
11.52½ CO_2 stopped. E.11%. Automatic breathing.
11.53 E.13%; CO_2 10%; O_2 26.5%. Blood pressure 180/100.
11.55 E. reduced 6% (to anticipate initial overdose); CO_2 10% O_2 25%.
11.56 E. 8%; CO_2 7%; O_2 29.5%. Fully anæsthetized.
12. noon Resp. 40; pulse 96; blood pressure 120/90.
12.7 E. 7%; CO_2 5%; O_2 31.5%.
12.10 Blood pressure 130/95; resp. 40; pulse 110. E. 7%; CO_2 5%; O_2 31.5.

12.15 Blood pressure 125/90; resp. 40; pulse 112. Percentage as before.
12.16 Anæsthetic stopped. O_2 4 litres; CO_2 2 litres. Respons CO_2 in 25 seconds.
12.17 Corneal reflex present. Blood pressure 120/90.
12.19 Brisk corneal reflex and cough.
12.21 Expiratory phonation. Blood pressure 130/110.
1.25 In ward, crying, able to talk and answer questions.

Comment.

Blood pressure higher throughout the administration than before.

The Inhaler and Table have been used in a large number of administrations, both in hospital and private practice.

A surgeon states "The clinical results are far in advance of anything previously attained: the control of the respiratory excursion at request is especially helpful and the muscular relaxation is perfect." A gynæcologist states "At the end of an hour's administration the patient is suffering little if at all from the anæsthetic: she may be suffering from the shock of the operation, but not from the anæsthetic; this is of the greatest importance in a long operation."

The following extract is made from a patient's letter dated the 12th November, 1928.

> "I felt you place a pad of wool soaked in Eau-de-Cologne over my mouth and nose. I took three or four breaths (not more) without the slightest discomfort and remember nothing more until I found myself in bed after my operation. One of the greatest advantages of your anæsthetic was there was no sickness afterwards. I never even felt sick. Before, when taking an anæsthetic I have always choked, and had a feeling of suffocation and afterwards been very sick."

ACKNOWLEDGMENT.

The Inhaler and Table have been beautifully made by Messrs. Mayer & Phelps, who have spared no pains in perfecting their manufacture.

The Flow-meters have been made by Messrs. Siebe, Corman & Co. Ltd.

MORTON BUST PRESENTATION ADDRESS.*

By F. H. McMechan, A.M., M.D.,

Avon Lake, Ohio.

THE world has many feasts and holidays, but among them there is none that approaches in interest or importance the one we celebrate to-day, and in commemorating "Ether Day" we look back upon that momentous occasion which initiated the era of painless surgery and we try to pay our respects and show our appreciation to Morton, the genius, who in this very amphitheatre, 82 years ago, for the first time publicly and successfully demonstrated the possibility and value of etherization.

It is easy enough to conceive a new idea in the study and work out its details in the laboratory, but to demonstrate its utility to a doubting public and a sceptical profession is quite another matter. Yet that is what Morton did and we cannot but admire him for his accomplishment, because in so doing he gave that gift of gifts—*painless surgery*—to suffering humanity, which had been awaiting this beneficence for untold ages and generations.

I cannot challenge your attention more dramatically to the value of anæsthesia than to ask you—"What would happen to-morrow if anæsthesia were blotted from the world to-day?" You can realise that the whole structure of modern surgery, a great part of medical practice and most laboratory research would go crashing into the abyss of oblivion. You cannot conceive of life going on without surcease from pain in face of the fact that, day by day, as many casualties of peace drift

* Address delivered on "Ether Day," on the occasion of the presentation of a bronze bust of W. T. G. Morton to the Massachusetts General Hospital for the Bullfinch Amphitheatre, where etherization for painless surgery was first publicly demonstrated. This bust was the gift of members of the Associated Anæsthetists of the United States and Canada and the International Anæsthesia Research Society.

into our hospitals as were evacuated from the trenches in the world war.

Imagine if you can the emotions of patients who had to endure the ordeal of a surgical procedure without anæsthesia and the dauntless courage of surgeons in performing their operations without relief of pain. Before Morton's demonstration, hospitals were looked upon as houses of terror and torture and death itself seemed preferable to the agonies of the operating room. We can scarcely credit that such conditions and circumstances ever existed.

We think of anæsthesia as having permeated the known world, and yet among nations of hundreds of millions of souls, painless surgery is as primitive now as it was among the Scythians and Incas and in China in the days of Hoa-tho. Even in our midst, how many countless things are done in our hospitals, clinics, offices and in the homes of our patients with never a thought of pain relief? This is one of the remaining cruelties of medical practice that must pass before we are really entitled to the affection and esteem of the public, which looks to us for delivery from unnecessary suffering.

In his day Morton flung us a torch, lit with the fire of his vision and burning wtih the inspiration of his ideals! In full confidence, I think I may say that this torch—the heritage of future generations of anæsthetists—was caught and has been carried by them proudly and with a full measure of achievement. Anæsthetists were not long in learning the lesson that for anæsthesia to achieve its full purposes, its organization must of necessity become world wide in scope and results.

Already the English-speaking world has accomplished its organization of the specialty. We have the Sections of Anæsthetics of the Royal Society of Medicine and the British Medical Association and a Section of Anæsthesia will be added to the Australasian Medical Congress at the meeting in Sydney, September, 1929. We also have the Scottish Society of Anæsthetists and the Associated Anæsthetists of the United States and Canada with its five reginal societies—the Canadian, Eastern, Mid-Western, Southern and Pacific Coast. All those have had a potent influence upon the profession at large for better anæsthesia not only through their own meet-

ings and programmes, but also through joint sessions with larger and more important medical, dental and surgical societies.

The most recent advance in organization is represented by the International Anæsthesia Research Society, which is attempting not only to foster anæsthesia research and the publication of the literature of anæsthesia, but also international contacts and fellowship. Its publication of *Current Researches in Anæsthesia and Analgesia* has been followed by the founding and publication of the *British Journal of Anæsthesia* and the two new German Journals of Anæsthesia —*Der Schmerz* and *Narkose und Anæsthesie*. These publications now stand as broadcasting stations for sending the latest and best knowledge of anæsthesia throughout the world and making this information available for study and further research and teaching purposes.

There is no phase of the practice of medicine which has closer connections with the basic biological sciences than anæsthesia. The activities of the Research Society have brought the achievements of pure science into the operating room and have sent the clinical problems of anæsthesia back to the laboratory for solution. To-day we find this amalgamation of correlated interests between science and practice in the annual Congresses of Anæsthetists and that fact adds so much more hope for the future of anæsthesia as a specialty in the practice of medicine.

The success and progress of any specialty has always rested on a foundation of experimental science as applied to clinical practice; and in visioning the future of anæsthesia, we cannot but feel convinced that the coming generation of anæsthetists must have all the resources of applied mechanics, physics, chemistry, bio-chemistry, pharmacology, physiology, and pathology as part of the basis of their future careers. Coupled with these there must also be a well rounded-out medical education in the essentials of the general practice of medicine and its specialties, and most particularly in the technical and clinical phases of anæsthesia.

From this we can see at once the necessity for every medical school becoming a centre for the development of

anæsthetists, beginning with instruction and animal experimentation in the basic science laboratories, continuing throughout the entire medical course for diagnosis, therapy and prognosis; and concluding with intensive experience for perfection in the specialty itself. More than half of the medical schools of the United States and all of those of Canada are already lending anæsthesia their whole-hearted co-operation in the teaching and instruction of anæsthesia through highly developed departments with opportunities for fellowships in the specialty. Their co-operation is certainly a challenge to those medical schools which are still lagging behind in this respect and are not keeping faith either with their students or the public.

If the medical profession is ever to accomplish the broader possibilities of anæsthesia in relation to public service, every physician must be qualified to give safely, pleasantly and efficiently such anæsthesia as may be required in painless childbirth, or minor surgery, whether in the home, industrial clinic, or office. Also a certain number of better trained and more competent medical anæsthetists are required to go into this work in the hospitals of the smaller towns, where more and more of the surgery of the future is going to be done. Teaching hospitals and medical schools must develop such professional anæsthetists as will, in didactic lectures, practical instruction and experimental research, vie in calibre with the heads of any other departments.

The general practitioner anæsthetist must fulfil the obligation of the profession to the public for the universal demand for the relief of pain. The clinical medical anæsthetist must be the internist of the surgical team and bring all the resources of the practice of medicine to the safety and recovery of operative patients. The teaching anæsthetist must be the liason officer between the research laboratory, the medical school and the surgical departments of the hospital. Organized anæsthesia looks forward to the day when all physicians will be able to fulfil the ideals of anæsthetic service; when all hospitals will have highly organized and proficient departments of anæsthesia and all medical schools chairs of anæsthesia.

The International Anæsthesia Research Society, has realised that in co-operating with the fourth, or physiological

era of surgery, the necessity of developing a safety-first campaign for preventing needless deaths and for bringing medicine from under the odium of the time worn adage—"The operation was a success but the patient died!" To accomplish this purpose, its Record Committee has developed a "safety-first" anæsthesia chart, which demands the evaluation of risk before operation, five-minute blood pressure guides and safeguards during the entire operative period and the determining of vital reserve depression through recovery indices for post-operative therapy and care. By the use of the "safety-first" chart the "supposedly fit" will cease to pass away unexpectedly and without adequate explanation; "the gravest risks" will no longer be hurried into operations without life-saving preparation and there will be no guess work about what is happening to the patient during the operative period. Finally, the critical 72 hours after operation, as yet so full of anxiety and so pregnant with surgical morbidity and mortality, will give way to the comfort, assurance and blessing of uneventful recoveries.

The Record Committee has also developed a "teaching" anæsthesia chart which enables students and internes to collate all pertinent data from the patient's history as well as the results of further indicated examinations and tests available for exhuastive pre-operative evaluation, operative protection and post-operative recovery; not to mention the charting of all the essential reactions to anæsthesia and surgery during the operative period itself for further study in bettering anæsthesia and surgery.

Organized anæsthesia also looks forward to the day when students and teachers of anæsthesia will be exchanged between the various countries in the international conference and it is hoped that, from time to time, the best representative of the specialty will come to present their latest ideas and practices to the Congresses of Anæsthetists, wherever they may be held, and that groups of anæsthetists will visit their confreres to see for themselves their clinical demonstrations.

While research workers have discovered and perfected newer general and local anæsthetics; and manufacturers have made them available in quantity and quality and with proper equipment for widespread use; and while anæsthetists and

others specialists have given them rapid and thorough clinical trial, the end is not yet. Perhaps even in our own day the very mechanism of anæsthesia itself may be explained and after that the possibilities for progress would seem to be limitless.

Almost every phase of industry and science has been abundantly fostered and financed by philanthropy. Seemingly only anæsthesia has been overlooked and neglected, and yet in the practice of medicine it holds out infinite possibilities for relieving suffering humanity. May I then appeal for the creation of its applications. The philanthropist who will confer this bounty in behalf of suffering humanity will need no recognition or reward for he and his kindness will be enshrined in the hearts of a grateful people forever.

In closing, I hope and trust, that you will now appreciate that the torch which Morton flung to us so many years ago has been carried through the generations since his time with credit and progress. To-day we have come to lay our achievements at his feet in a reverend spirit of humility and modesty, but not without a certain feeling of pride and a sense of accomplishment.

This bust is presented in behalf of the Associated Anæsthetists of the United States and Canada and the International Anæsthesia Research Society to the Trustees of the Massachusetts General Hospital as a token of their esteem for the part which the hospital played in the development of painless surgery and in founding and continuing "Ether Day" so that the first public demonstration of etherization might be fittingly commemorated every year.

Showing Morton just as he was on the day he entered the Bullfinch amphitheatre and demonstrated to Warren, Bigelow and the others present, that painless surgery was an accomplished fact, we hope that this symbol of the man may remain, throughout the years to come, as a memorial to his glory and a recognition of what he accomplished; and that its presence here may make a shrine for all the anæsthetists of the world to come to and understand and learn the lesson which Morton taught.

AN EVALUATION OF PRELIMINARY INJECTIONS.

By E. Falkner Hill, M.B., Ch.B. (Vict.).
Lecturer in Anæsthetics, University of Manchester.

THIRTY years ago in Manchester all surgical operations were done under chloroform or ether. The chloroform was given on lint, and ether by means of Clover's ether inhaler. For about ten years these methods persisted unmodified. In 1908 or 1909 the Roth Drager Guglielminetti instrument was bought. This was an excellent apparatus for hospital work. By its means it became possible to administer a known and easily modified percentage of chloroform or ether or a mixture of the two, with oxygen. Of course it had its faults, faults common to nearly all instruments, it was expensive, liable to get out of order, and portable only with difficulty. For these reasons it was not long before it fell into disuse altogether. It was whilst using this particular machine that I saw a death on the operating table for the first time. The patient, a woman suffering from a malignant enlargement of the thyroid, died during the operation immediately after severe traction on the tissues of the neck. At the inquest subsequently held by the coroner, the verdict was brought in that death was due to natural causes accelerated by the anæsthetic. I think it would have been more correct to have said that death was due to natural causes, accelerated by the operation which the anæsthetist, unfortunately, was unable to prevent. Surely the factors causing this death were: firstly, the disease; secondly, the severe traction on the tissues of the neck; and thirdly, the anæsthetist was unable to prevent the disastrous effects of this combination. However, as a death on the table is a very regrettable incident whatever may be the immediate cause of it, it is one of the functions of the anæsthetist to prevent it if he can. After careful consideration of this case, and a helpful letter from Sir Clifford Allbutt, I decided to give atropine gr. 1/60 and morphia gr. ¼ in all cases in which there was any likelihood of shock, and, especially, in any cases in which there was any possibility of traction on the vagus. Within a year of this date at the invitation of Dr. Archibald Donald of Manchester, Dr. A. M

Mitchell and I went to Liverpool to see Dr. Grimsdale operate under "open ether." As everybody knows, an important feature of the open ether method is the preliminary injection of atropine. This method of anæsthetizing was there and then adopted in the Gynæcological Departments at the Manchester Royal Infirmary and at St. Mary's Hospital. It is a method particularly suitable for anæsthetizing women, in whom as a rule it is much simpler to obtain the desired degree of muscular relaxation than it is in men. In the course of the next few years the use of atropine as a preliminary injection to anæsthesia spread throughout the Manchester Royal Infirmary, and it has for many years now been the routine procedure. To my mind it is the most important advance in the practice of anæsthesia that has occurred in the last twenty years. It seems to me likely that there are hundreds of people, possibly thousands, now living who, but for its timely aid, would now be dead. Testimonials to its efficacy are not wanting. A nurse who, after four years private practice, returned as night Sister to one of our Manchester nursing homes, reported to the Matron that the chief difference she noticed in the patients was the extraordinary diminution in the vomiting. When this nurse was in the Home before her period of out-nursing, anæsthesia had been induced with ethyl chloride and ether; after her return, with ether only, preceded by atropine grain 1/60. How far the absence of ethyl chloride contributed to this desirable result I am unable to say, as my experience with this drug is too small. That atropine was the main factor is, I am sure, the opinion of those who have had the greatest experience with it. From the point of view of the patient's comfort then, this action of atropine in preventing secretion is of very great importance, but from the point of view of the patient's safety atropine has an action of even greater importance. It is scarcely too much to say that the preliminary injection of atropine has made $CHCL_3$ a safe anæsthetic. Let me say at once some of the things I don't mean. I don't mean that patients never die under an anæsthetic even if that anæsthetic is chloroform. That would be putting the claims of chloroform too high. Chloroform is not the elixir of life. Secondly, I do not mean that chloroform is fool-proof in the sense that any man with a medical qualification may safely administer

chloroform to any patient whatever his condition by merely giving the preliminary dose of atropine. Thirdly, I do not mean that when a patient is under chloroform it will prevent him dying of toxæmia, shock or hæmorrhage. It probably adds to an already existing toxæmia, but many people believe that it tends to lessen both shock and hæmorrhage. The habit of regarding all deaths that occur under anæsthesia as due to the anæsthetic is indirectly responsible for a number of deaths which would not otherwise occur.

During the last twenty years I have seen three cases of early collapse under chloroform; in all these cases atropine had been ordered as usual but it had not been given. I did not ascertain this fact until after they had recovered. If that is a coincidence it is a very remarkable one; for my part I put it down to the fact that these patients were not protected by atropine.

How does atropine act? No fact in physiology is better attested than the action of atropine on the branches of the vagus that run to the heart. Though it has no action on the pre-ganglionic fibres it paralyses the post-ganglionic terminals in the heart muscle and so allows the heart to escape the inhibitory influence of the vagus. I am aware that this is an old and time-honoured explanation but it is not, therefore, necessarily wrong. However, atropine has its critics. Thus Levy, in his book on chloroform anæsthesia, says "Its use has been extensively advocated as a prophylactic against sudden death from a presumed vagus inhibition of the heart, but in the light of our present knowledge of the pathology of sudden death it must be considered entirely useless in this connection." He adds in a foot-note that it may be useful at a later stage to restrain vagal action. Is it possible to harmonize these diametrically opposed views? I think it is. Levy regards death in the early stage of chloroform narcosis as due to ventricular fibrillation. Whatever may be the essential state of the heart muscle fibres in fibrillation, or delirium cordis as it has been so well named, there is no doubt that both the excitability and the conductivity of the heart muscle are depressed. Now, this condition of depressed excitability and conductivity is precisely the condition brought about by stimulation of the vagus. The conclusion is obvious.

If in two very important respects stimulation of the vagus brings about that condition of the heart muscle which is found in fibrillation, and atropine prevents stimulation, then atropine is very strongly indicated as a preliminary injection to chloroform narcosis. In fact, I would go further and say, if this reasoning is accepted, that chloroform should never be given unless atropine has been given first. That has been my practice in some 20,000 cases ever since I saw the case, to which I referred just now, die on the table. I see no reason to alter that practice.

What are the reasons for the preliminary injection of morphia? When I first started to give it I had two main objects in view, viz: one, the prevention of physical shock; and two, the prevention of psychical shock. To most people the thought of an operation is disturbing, to a few it is an occasion for panic. I feel convinced that these latter, by their excessive fear, place themselves in a position of considerable danger when they submit to an anæsthetic. One-fourth grain of morphia, an hour before the operation to these is not only a kind attention, it is a real safeguard. Few people are so self-possessed before operation that the soothing influence of morphia is other than a welcome relief.

With regard to the question of physical shock, whatever view you may take of the intimate pathology of shock, there is a condition with which every practical anæsthetist is familiar. Following some considerable injury to the patient such, for example, as severe traction on the spermatic cord, evisceration, packing three or four large swabs into the abdominal cavity, or even raising the bridge as in a gall-bladder operation, the radial pulse tends towards disappearance may in fact arrive at it. This is generally associated with some degree of cyanosis, quiet to shallow respiration, and, frequently, profuse perspiration. As this state of the patient is induced with great rapidity (a minute or two) it seems natural to suppose that it is a nervous phenomenon either in origin or in effect. Again, it is well-named "Shock," as the word is derived from the Anglo-Saxon word *scacan* which means to shake, and I think we should all agree that evisceration or any of the above mentioned proceedures would be likely to shake even the stoutest, in the absence of an

anæsthetic. Recently, such very different ideas as to the nature of shock and its cause have been put forward, e. g. that it is not due to the trauma but to the anæsthetic, that it is due to the transmission not of nervous impulses but of chemical poisons (histamine etc.), or, again, that it is due to a particular condition of the protein particles in the blood, that I thought it better to describe the state of the patient to whom I was referring, than merely to describe it as shock. It is this state that the prelimnary injection of morphia tends to prevent, or, at least, minimize. It is difficult to get away from the idea that the patient who has had morphia is better able to stand, both psychologically and physically, the inevitable injury which a major operation involves.

These then were the two main reasons which led to the preliminary injection of morphia. In practice I have seen nothing to shake my faith either in the reasons or their clinical application. Truth as we are all aware is many sided but one aspect of truth never contradicts another, rather does it confirm that other, and now we have apparently another example of such a confirmation. During the last few years the question of the CO_2 content of the alveolar air and the blood has been brought prominently before the notice of the profession, notably by Yandell Henderson. Henderson and Haggard have shown how the hyperpnœa associated with numerous conditions, of which severe visceral manipulations as in laparotomy is one, may lead to a fatal apnœa by the acapnia resulting from the excessive elimination of CO_2. Now morphia directly depresses the R. C. It is in fact the leading argument of the opponents of morphia in this connection, and so prevents the hyperpnœa which usually results from these excessive stimulations and consequently the acapnia and all the disorders of the R. C. and V. M. systems associated with acapnia. Here, then, is another strong argument for morphia as a preliminary injection, and not for morphia alone but for some chloroform and some ether rather than a large quantity of the latter with its tendency to produce hyperpnœa, acapnia and its attendant mischiefs. The depression of the R. C. then as caused by morphia turns out apparently to be another of the advantages, rather than one of the disadvantages, of the preliminary injection of morphia.

INVESTIGATIONS OF THE CHEMICAL CHANGES IN METABOLISM DURING ANÆSTHESIA.*

By Florence Stephen, M.B., Ch.B., L.R.C.P., M.R.C.S., M.Sc.

(*The British Journal of Anæsthesia* offered a prize of Fifty Pounds in memory of the late Dr. S. R. Wilson, for the Best Inhalation Anæsthesia Research Effort by a Citizen of the British Empire in 1928. The judges decided the following to be worthy of the Award).

Continued from the January Number

Theories of Origin of Increase in Neutral Sulphur after Anæsthesia.

The following suggestions are based on the theory of Warburg,[87] of tissue respiration, and the work of Hopkins and others[88] on glutathione.

Warburg's theory.

As a result of his experiments on the adsorption of amino-acids by charcoal and his discovery that the subsequent oxidation proceeds to the same products as in the body at body temperature, Warburg has shown that in oxidative processes in cell metabolism the substance to be oxidised is adsorbed on the surface of structural elements of the cytoplasm, and he believes that these oxidative or respiratory processes are carried out on the surface of solid phases of the cell structure, and are catalysed by iron, since, (1) all oxygen consumption ceases if cells are mechanically dis-

*This research was commenced at the instigation of the late Dr. Sidney Rawson Wilson, who suggested to the writer that there were many chemical problems in anæsthesia which required investigation.

Unfortunately the writer was not able to discuss these problems in any detail with Doctor Wilson before the latter's so unfortunate accident occurred, so that the work has been continued alone.

The investigations pursued have extended over a wide field, and the results can best be estimated by references to the Summary at the conclusion.

integrated, while killing cells with acetone allows some oxygen to be used since the structure is unchanged. (2) The oxidation of lecithin proceeds in presence of iron, but lecithin alone takes up no oxygen (Thunberg[89]).

Glutathione.

Hopkins and his co-workers have isolated a substance called glutathione, which they regard as one of the main factors in tissue oxidation.

This substance is a compound of glutamic acid and cystein and exists in two forms:—(*a*) a reduced form; (*b*) an oxidised form.

It is noteworthy that cystein is characterized by the ease with which it is converted into the oxidised form, cystine, and under certain conditions the reverse process may be accomplished, this auto-oxidisability being catalysed by the presence of small amounts of iron. There is no evidence that either cystine or cystein occur in the tissues, although actively metabolic tissues give the intraprusside reaction characteristic of cystein: the substance giving the reaction in this case having been proved by Hopkins to be glutathione.

Glutathione probably corresponds to the "Pnein" of Batelli and Stern,[90] which was shown to influence the respiration of muscles, and as such, therefore, it must occur on the surface of the cell, for it may be washed off easily. It appears to act in conjunction with a substance in the tissues which is thermostable and resists heating to 100°C. (Hopkins and Dixon[91]): it occurs in relatively large amounts in glands and has been identified as a constituent of the red corpuscles (Holden) and accounts for much of the neutral sulphur of the blood.

It may be assumed then that oxidation in the animal tissues proceeds by union of oxygen with the cell membrane in presence of certain catalysts and an intermediate substance, i.e., iron and glutathione based on the foundation of a peroxide available for tissue oxidation:

Glutathione + peroxidase – oxidase cystein – peroxides
(Thurlow,[92] B. J., 1925, 19, 199).

Warburg has shown that narcotics depress the respiratory activity of the cells by displacing oxidisable substance from the surface of the cell, and the normal metabolism of the cell

cannot proceed, the amount of depression of cell oxidation being proportional to the degree of absorption of the narcotic. This theory may serve to explain the mechanism of anæsthesia, and it is therefore suggested that the anæsthetics displace glutathione from the surface of the cells so that this substance becomes released from the tissues where it is present in the reduced form and enters the circulation, being carried to the liver. It is suggested, further, that the glutathione is decomposed into glutamic acid and cystein, which becomes oxidised to cystine, which is then excreted in the urine unchanged or at least undergoes a modified degree of oxidation in the liver, so that some of the sulphur is excreted in an unoxidised form as neutral sulphur. Another source of glutathione may be as a result of hæmolysis which Webster[93] states occurs during anæsthesia.

It is obvious, as previously stated, since ether is more widely diffused through the tissues than chloroform and produces a recognisable depression in tissue oxidation (McKesson[94]), then accordingly the action of ether in displacing glutathione from the cells will be more marked, and hence the excretion of neutral sulphur should be higher after ether anæsthesia than after chloroform, which has been shown experimentally to be the case, so that this fact lends some support to the above theory of anæsthetic action.

It is interesting to note that in 1904 Moore and Roaf[95] suggested that anæsthetics were absorbed by all proteins with consequent limitation of the activity of "bioplasm" and its power of combination with bodies for metabolism.

The importance of sulphur in metabolism was early recognised and its relation to the oxidation processes of cells since, as pointed out by Blanchetiere[96] and later, Flurin[97], it has a profound oxid-reducing property, and the SH bodies are recognised as possessing anti-toxic powers.

Further it may be noted that Loeper[98] has found that neutral sulphur is almost exclusively retained by the suprarenals, and that accumulation of it is associated with accumulation of amines, and, in relation to anæsthesia, it is possible that the facts may in some way be linked up with the phenomenon observed by Delbet, that the fat of the suprarenal gland and the chromaffin substance "fixed" the narcotic

to such a degree that the content of the capsule becomes greater than that of the blood, and further that the fat of the zona fasciculata tends to extend into the zona mediata in proportion to the duration of anæsthesia. In relation to this latter statement Nicloux and Yocanovitch[99] found $CHCl_3$ occurs in largest extent in fatty tissues, and Reicher[100] states that important lipoids and fats are expelled by cells under anæsthesia and may play an important part in the production of anæsthesia.

Albumenuria.

Terrier[101] in 1884 reported the occurrence of albumenuria after chloroform anæsthesia, this being later confirmed by Eisendrath,[102] for ether anæsthesia also, but more frequently after chloroform. Zachrissen,[103] Babachi and Bebi[104] also found albumen in post-anæsthetic urine and the former suggested the extent of albumenuria was proportional to the length of the operation.

In 1899 and 1900 theories were put forward by Kemp and Thompson[105] and by Buxton and Levy,[106] respectively, to account for the albumenuria after ether anæsthesia. It had been generally accepted that the albumenuria after anæsthesia was a result of the effect of the drug, either chloroform or ether, on the organs. Kemp and Thomson, and later Buxton and Levy recognised the importance of the direct action of ether on the renal arteries in producing contraction of these and ischæmia of the kidneys with damage to renal epithelium and secretions, and consequent albumenuria. Bovee[107] observed also the influence of the temperature and fall in blood pressure during narcosis, and drew an analogy with the "specific renal effect" of Nuszbaum produced by prolonged contraction of renal arterioles and the kidneys in the frog after ligature of the renal arteries.

Grondahl[108] found albumenuria in 38 per cent. of ether cases and 18.5 per cent. chloroform cases, the condition lasting for from seven to nine days according to the age of the subject, time of operation and amount of anæsthetic used. Bovee, and later Schulze,[109] found transient albumenuria after ether also. The question was reviewed by Haines and Millikin[110] in 1927 and it was suggested that ether acts as a

vasomotor stimulant and causes constriction of the vessels: this gives rise to diminution in the output of urine, anuria and ischæmia of the kidneys with secondary capillary hyperæmia anuria, serum transudate and albumenuria.

From Koehler's[111] theory the conditions of acidosis—ketosis produced in anæsthesia might be expected to have some influence on the condition of post-anæsthetic albumenuria, since he considers that certain renal changes occur as a result of the depression of tissue oxidation in ketosis, but Bjure[112] suggests an analogy between albuminuria after anæsthesia and that frequently seen in diabetes and occurring with only slight ketosis.

In the 60-70 cases examined, all those in which ether had been administered showed albumen in the post-operative urine, while in the chloroform cases albumen was present in five cases. The condition cleared up in about three days after ether, but albumen was present in one post-chloroform urine on the fifth day after operation. No relationships were apparent between the degree of the albumenuria and the length of operation or amount of anæsthetic. It would appear, therefore, that the ether has a constant effect on the kidneys in producing transient renal damage, probably slight cloudy swelling and albumenuria, which may be due to two factors:—

1. The effect of the anæsthetic, i.e., ether acting as a vasomotor stimulant and producing ischæmia of the organs as suggested by Haines and Millikin.

2. The greater depression of tissue oxidation which, it is suggested previously, may be produced by ether—this also producing changes in the renal cells.

Urobilinuria.

The presence of urobilin in post-anæsthetic urine was first noticed by Kast in 1888, his observation being supported by Chiarleoni[113] and others, but it does not appear from investigation of the literature that any quantitative estimations have yet been made.

Several urines have been examined by the fluorescene test for urobilin and a definite difference in the degree of fluorescence between the pre- and post-anæsthetic urines

observed. The quantitative relations of the extent of urobilinuria before and after anæsthesia as a test of liver function as suggested by Addis,[114] will shortly be investigated.

Nitrogen Excretion after Anæsthesia.

Straszmann, in 1880, reported an increased nitrogen excretion in starved dogs after chloroform anæsthesia and attributed it to increased protein breakdown. His results were not confirmed entirely, however, by Thompson,[115] who found a decrease in total nitrogen after chloroform, and a diminution in percentage of nitrogen in the urine after ether and chloroform with variations in the case of anæthetic mixtures. A little later Pechell[116] found a decrease in nitrogen immediately after anæsthesia with an increase on the subsequent days, and also a tendency for the urea to decrease, and finally considered from these and other observations that there is a profound change in composition of the urine after anæsthesia, and nitrogen is excreted in other forms than urea, uric acid and ammonia. He also drew an interesting analogy between anæsthesia and exhausting work when out of condition, since each condition is followed by an increase in urea and total N_2 excretion, also uric acid and xanthins.

Further investigations by Pringle and Maunsell[117] showed a decrease in nitrogen excretion, which they stated amounted to practical suppression due to inactivity of renal epithelium. Thompson[118] published some further researches which gave further support to his own and the later work, and stated that a greater arrest of function occurs with ether than with chloroform.

About ten cases were investigated and the results obtained agreed with those given above, .e., there is a diminution in nitrogen and the urea excretion, but an increase in the ammonia and amino-acids (taken together) and also in the acidity of the urine. These latter have already been investigated by Reimann and Hartmann[119] and Stehle, Baume and Barbour[120], although Collip[121] found no increase in acidity. Stehle and Baume[122] acount for the increased acidity as due to increased excretion of phosphoric acid which they proved experimentally; this was attributed to them and by Ronzoni[123] to discharge of phosphorous from muscles as a result of

breakdown of the hexose phophate compound—i.e., lactacidogen postulated by Embden.[124] Such a phenomenon would also account for the increase in lactic acid after anæsthesia.

NITROGEN EXCRETION DURING ANÆSTHESIA.

No.	Acidity A	Acidity B	Amino acids A	Amino acids B	Nitrogen A	Nitrogen B	Operation	A.D.	Mins.
1	191	915	.2183	.3577	1.201	.413	Appendix.	E.	45½
2	131.9	737.5	.1832	.2115	1.536	1.217	,,	E.	60
3	47.7	421.5	.0215	.13	.8072	1.34	,,	E.	50½
4	85.4	478	.0525	.1164	.3618	.3221	,,	E.	40½
					Urea.				
					.1458	.4613			
9	95.2	247.5	.0385	.0525	.5423	.4203	Cholecystectomy.	E.	55
10	59.1	93.3	.0833	.3139	1.43	1.269	I.D.K.	E.	50
					Urea.				
					1.504	2.203			
11	152.5	103.0	.0767	.2124	.4616	.8716	Laparatomy.	E.	40
12	257.2	464	.102	.139	1.170	.9434	D.I.H.	E.	60
					Urea.				
					.1123	.3077			
13	212.4	336.4	.0403	.1155	.2999	.1060	Nephrectomy.	E.	60
					Ureq.				
					.7726	1.19			
22			.1176	.1457	.9145	.4982	Cholecyst.	E.	45½

Values in mg. per 100 c.c.
N.B. A=before anæsthesia.
B=after ,,

SUMMARY.

Investigations have been carried out on the following lines:—

1. On TOXÆMIAS as evidenced by acetonuria and glycosuria:

(*a*) Post-anæsthetic acetonuria has been found to occur chiefly in nervous patients, and a suggestion has been put forward that its presence may be accounted for by endocrine disturbance, or by the diminution of oxidation produced by anæsthetics.

(*b*) An increase of reducing power of the urine has been noted after anæsthesia, which may be due to glycosuria or may have some relation to the increased post-anæsthetic output of urochrome.

2. On CREATININE and CREATINE EXCRETION :—

(*a*) An increase in percentage of creatine excretion has been observed to occur after anæsthesia, while that of the creatinine appears to vary.

(*b*) An increase in the creatine-creatinine ratio after ether and chloroform anæsthesia has been observed, showing that ether also produces changes in the liver but of a milder nature than those produced by chloroform.

3. On NEUTRAL SULPHUR EXCRETION after anæsthesia :—

(*a*) This has been found to increase in both percentage and total values.

for any one operation, but depends upon the anæsthetic,

(*b*) The increase appears to be the same on an average being higher for ether than for chloroform.

(*c*) The excretion of cystine and also urochrome have been found to increase after operation.

(*d*) It is suggested that the increase in neutral sulphur and in cystine is related to the "glutathione" oxidation mechanism of cellular metabolism.

4. On ALBUMENURIA :—

This condition has been found to occur after ether anæsthesia in all cases examined and after chloroform anæsthesia in 20 per cent. of cases.

5. On UROBILIN :—

A quantitative increase in urobilin excretion after anæsthesia has been observed.

6. On NITROGEN EXCRETION after anæsthesia :—

The nitrogen excretion etc. have been examined in 9-10 cases and the following results obtained :—

1. Increase in acidity of the urine after anæsthesia.
2. Increase in percentage ammonia and animo acids as estimated together by the formal titration.
3. Decrease in percentage total nitrogen excretion after anæsthesia.
4. Increase in percentage urea excretion after anæsthesia.

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BIBLIOGRAPHY.

Introduction.

1. Caspar. *Klin. Wochenschrift.* 1, 1, 1850.
2. Heintz. *Inaug. discussion Rotterdam,* 1896.
3. Bandler. *Mittelteil. aus d. Grenzgebeit,* 1896. *Eulenbergs Vierteljahr,* 47.
4. Ungar. *Mittelteil. aus d. Grenzgebeit,* 1896. *Eulenbergs Vierteljahr,* 47.
5. Junkers. *Mittelteil. aus d. Grenzgebeit,* 1896. *Eulenbergs Vierteljahr,* 47.
6. Ostertag. *Virchows Arch.,* 118, 2.
7. Nothnagel. *Berliner Klin. Woch.,* 1896.
8. Guthrie. *Lancet,* II, 1903.
9. Goodhart. *Brit. Med. Journ.,* II, 1910.
10. Graham. *Journ. Exp. Med.,* 22, 48, 1915.
11. Reichel. *Archiv. f. Klin. Chir.,* 135, 1925.
12. Kast. *Zeit. f. Klin. Med.,* 1888.
13. Zeller. Quoted in *Scott. Med. Journ.,* 1904.
14. Kast and Mester. *Zeit. f. Klin. Med.,* 18, 1891.
15. Grube. *Jahrbuch d. Chir.,* 1899.
16. Chiarleoni. *Jahrbuch d. Chir.,* 1899.
17. Strazzmann. *Virchows. Arch.,* 115.

Toxæmia.

18. Becker. *Virchows Arch.,* 90. *Deut. Med. Woch.,* 16, 1894; 18, 1895.
19. Luzatti. Quoted in *Scott. Med. Journ.,* 1904.
20. Hill Abram. *Journ. Path. and Bact.,* 1896.
21. Nachod. *Archiv. f. Klin. Med.,* 50, 1895.
22. Waldvogel. *Langenbecks Archiv.,* 66, 1902.
23. Stiles and McDonald. *Scott. Med. Journ.,* 1904.
24. Stone and Law. *Boston Med. Journ.,* 1903.
25. Kelly. *Ann. Surg.,* 1905.
26. Beesley. *Brit. Med. Journ.,* 1906.
27. Young and Williams. *Boston Med. Journ.,* I, 1908.
28. Apperly. *Brit. Med. Journ.,* II, 264, 1912.
29. Reimann and Bloom. *Journ. Biol. Chem.,* 36, 211, 1918.
30. Short. *Journ. Biol. Chem.,* 41, 503, 1919.
31. Grosz. *Brit. Journ. Anæs.,* 2, 1924.
32. Leake. *Brit. Journ. Anæs.,* 2, 1924.
33. Atkinson and Ets. *Journ. Biol. Chem.,* 52, 5, 1922.
34. McNider. *Anæs. and Anal.,* Aug., 1926.
35. Burge and Estes. *Anæs. and Anal.,* 34, 1928.
36. Perry and Thalhimer. *Journ. Am. Med. Ass.,* 81, 1923.
37. Caldwell and Cleveland. *Surg. Gyn. and Obstet.,* 1925.
38. Schulze. *Zeit. f. Chir.,* 51, 1924.
39. Waller. *Zeit. f. Gynak.,* 25, 9, 1926.
40. Rabinowitz. *Russkaia Klin. Moscow,* 1925.
41. Levy. *Deut. Med. Woch.,* 53, 1927.
42. Steigemann and Jugatti. *Med. Klin. Berlin,* 1925.
43. Cannon. *Journ. Am. Med. Ass.,* 78, 1922.
44. Ogawa. *Beitrage d. Physiol.,* 3, 134, 1925.
45. Zuntz and Barre. *Comp. Rend.,* 98, 858, 1928.
46. Burge and Estes. *Anæs. and Anal.,* 34, 1928.
47. Delbet. *Journ. An. Med. Ass.,* 1925.
48. Gougerot and Peyr. *Comp. Rend.,* 93, 1925.

49. Maranon. *Press. Med.*, 33, 1925.
50. Thompson. *Brit. Med. Journ.*, 1900.
51. King. *Bull. Johns Hopkins Hosp.*, 1904.
52. Moore and Roaf. *Proc. Roy. Soc.*, 73, 1904; 74, 1905.
53. Edie. *Biochem. Journ.*, 1, 1906.
54. Hawk. *Amer. Journ. Physiol.*, 10, 1904.
55. Ross and Keeton. *Amer. Journ. Physiol.*, 48, 1919.
56. Ross and Davis. *Amer. Journ. Physiol.*, 53.
57. Leake and Hertzmann. *Journ. Amer. Med. Ass.*, 82, 1924.
58. Minnitt. *Lancet*, 1, 761, 1925.
59. Pemberton and Cunningham. *Lancet*, 1924.
60. Tatum and Atkinson. *Journ. Biol. Chem.*, 54, 1922.
61. Josephs. *Bull. Johns Hopkins Hosp.*, 1925.
62. Foshay and Boyd. *Archives of Surg.*, Sept., 1927.

Creatinine.

63. Folin. *Amer. Journ. Physiol.*, 13, 1905.
64. Shaffer. *Amer. Journ. Physiol.*, 23, 1908.
65. Steinbock and Grosz. *Journ. Biol. Chem.*, 36, 1918.
66. Underhill. *Journ. Biol. Chem.*, 27; 127; 1916.
67. Steinbock and Grosz. *Journ. Biol. Chem.*, 36, 1918.
68. Denis and Minot. *Journ. Biol. Chem.*, 37, 1919.
69. Marongni. *Palioclinico*, 33, 619, 1926.
70. Folin. *Journ. Biol. Chem..*, 17, 1914.
71. Wolf and Osterberg. *Phys. Rev.*, 1924.
72. Boshamer. *Klin. Woch.*, March, 1928.
73. Whipple and Speed. *Journ. Exp. Med.*, 21, 3.
74. Rosenthal and Bourne. *Anæs. and Anal.*, 1928.

Sulphur.

75. Dombrowski. *Zeit. f. Physiol. Chem.*, 54.
76. Lepine. *Comp Rend.*, 91.
77. W. Smith Jerome. *Pflugers. Arch.*, 60.
78. Burge and Grauhan. *Klin. Woch.*, 36 and 37, 1927.
79. Rosenheim and Drummond. *Biochem. Journ.*, 8, 134, 1913.
80. Samson Wright. "Applied Physiology," S. Wright.
81. Yandell Henderson. "Applied Physiology," S. Wright.
82. Baumann and Goldman. *Zeit. f. Physiol. Chem.*, 12.
83. Gawinski. *Zeit. f. Physiol. Chem.*, 58.
84. Rowe and Proctor. *Journ.. Biol. Chem.*, 4, 74, 1927.
85. Drabkin. *Journ. Biol. Chem.*, 443, 1927.
86. Folin and Looney. *Journ. Biol Chem.*, 51, 427, 1922.
87. Warburg. *Ergebnisse d. Physiol.*, 14, 1914. *Bioch. Zeit.*, 119, 1921; 142, 1923.
88. Hopkins. *Biochem. Journ.*, 15, 1921; 18, 1924; 19, 1925.
89. Thunberg. *Zeit. f. Physiol.*, 23, 1909. *Skand. Arch. and Physiol.* 1910.
90. Batelli and Stern. *Biochem. Zeit.*, 30, 172, 1910.
91. Hopkins and Dixon. *Journ. Biol. Chem.*, 55, 1922.
92. Thurlow. *Biochem. Journ.*, 19, 199, 1925.
93. Webster. *Amer. Journ. Surg.*, 105, 1921.
94. McKesson and Clement. *Brit. Journ. Anæs.*, 23, 1925.
95. Moor and Roaf. *Proc. Roy. Soc.*, 73, 1904; 73, 1905.
96. Blanchetiere. *Prog. Med.*, 53, 1926.
97. Flurin. *Prog. Med.*, 53, 1906.

98. Loeper. *Prog. Med.*, 53, 1901.
99. Nicloux and Yovanovitch. *Comp. Rend.*, 93, 1926.
100. Reicher. *Zeit. f. Klin. Med.*, 65.

Albumenuria.
101. Terrier. *Bull. Soc. Chir.*, 1884.
102. Eisendrath. *Zeit. f. Chir.*, 40, 1895.
103. Zachrissen. *Jahr. f. Chir.*, 1896.
104. Babachi and Bebi. *Jahr. f. Chir.*, 1896.
105. Kemp and Thompson. *New York Med. Journ.*, 1899.
106. Buxton and Levy. *Brit. Med. Journ.*, 1900.
107. Bovee. *Amer. Journ. Obstet. and Gynecol.*, 1909.
108. Grondahl. *Duet. Med. Woch.*, 25, 1905.
109. Schulze. *Arch. f. Klin. Chir.*, 139, 1926.
110. Haines and Milliken. *Journ. Urology*, 17, 1927.
111. Kœhler. *Journ Biol. Chem.*, 72, 99, 1927.
112. Bjure. *Upsala Lokare forenings Fordhandlingar*, 10, 9, 1926.
Urobilinuria.
113. Chiarleoni. *Jahr. f. Chir.*, 1899.
114. Addis. *Arch. Int. Med.*, 15, 1915.

Nitrogen.
115. Thompson. *Brit. Med. Journ.*, 1900.
116. Pechell. *Brit. Med. Journ.*, 1903.
117. Pringell and Mansell. *Brit. Med. Journ.*, 1905.
118. Thompson. *Brit. Med. Journ.*, 1906.
119. Reimann and Hartmann. *Amer. Journ. Physiol.*, 50, 82, 1919.
120. Stehle, Bourne and Barbour. *Journ. Biol. Chem.*, 53, 341, 1922.
121. Collip. *Brit. Journ. Exp. Path.*, 1, 282.
122. Stehle and Bourne. *Journ. Amer. Med. Ass.*, 83, 117, 1924.
123. Ronzoni. *Journ. Biol. Chem.*, 61, 1924.
124. Embden. *Zeit. f. Physiol. Chem.*, 113, 1921.

The Mayo Clinic announce the establishment of a Fellowship in Anæsthesia at the University of Minnesota. Particulars may be obtained by addressing Section of Anæsthesia, Mayo Clinic, Rochester, Minnesota, U.S.A. or to the Editor of the British Journal of Anæsthesia.

PRESSE MEDICALE, 17 OCT., 1928.

By M. M. Forgue and Basset.

Extract of an exhaustive paper on spinal analgesia read by M. M. Forgue of Montpellier, and Basset of Paris, and discussed by a large number of authorities at the thirty-seventh meeting of the French Surgical Association, Oct., 1928.

In order to appreciate the indications for spinal anæsthesia with its technique, limits, and dangers, we must try to understand its mechanism:—

In the first place it is important to distinguish between the puncture of the theca and the injection of the anæsthetic drug.

A simple puncture, even with a very fine needle, leaves a wound of the dura through which fluid may continue to escape after the needle has been withdrawn, a possible cause of headache and nausea, especially if any considerable quantity of fluid has been drawn off at the time of puncture; Pech and Delmas have shewn that a low tension in the C.S. fluid favours diffusion of the anæsthetic, but Forgue protests against the custom of withdrawing such quantities as 25 or 30 c.cm. The height to which analgesia will extend is a problem of bio-physics rather than of simple physics; by a selective action the drug becomes "fixed" to certain tissue-elements and the upward spread of its effect is limited by exhaustion of the reagent in solution. Another factor determining the limitation and distribution of analgesia is the affinity shown by the drug for the spinal roots, especially the posterior roots; the safety of this method is, moreover, enhanced by the superficial nature of the penetration of spinal cord tissues, as shewn by Gestan, Riser, and Laborde. As to changes produced in nerve elements, van Lier and Wossidlo found in rabbits and dogs that spinal anæsthesia produced lesions of the ganglion cells with disintegration of Nissl bodies but that these effects were transitory, complete recovery

taking place in 24 hours. As regards elimination of the drug it has been shewn by Klose and Vogt that this begins more slowly than might be expected from clinical data; Tropacocaine elimination commencing in 3 hours, that of novocaine in 12 hours, and of stovaine in 30 hours; the process being completed in from 20 to 48 hours, according to which drug is concerned.

The majority of workers regard spinal anæsthesia as too dangerous when employed as routine for upper-abdomen surgery, and although it may be indicated in order to avoid lung complications in such cases where local analgesia is insufficient, it is often safer, in gall-duct surgery, to supplement the local with open ether. The true domain of spinal is below the umbilicus, and it is generally agreed that where a short ethyl chloride or ether anæsthesia will suffice spinal introduces an unnecessary risk.

The property which spinal anæsthesia has of stimulating peristalsis renders it especially useful in certain caes of ileus and, on the other hand, may make its employment disastrous in operation for strangulated hernia owing to a risk of spontaneous reduction before the opening of the sac.

Among contra-indications must be included:—Subjects of low blood pressure, marked toxicity of obstruction; badly compensated heart disease; internal hæmorrhages; infection such as septicæmia, tubercle, or syphilis.

In summing up the authors compute the incidence of fatal accident at 1 in 1,000; of headache as 3 to 4 per cent; of 6th nerve paralysis 1 to 5 per thousand; and state that, far from unduly extending the field of spinal, they have learned to be more particular as to indications, and to widen the field of local and regional analgesia, returning to inhalation anæsthesia in short and simple cases.

During the subsequent discussion the following views were expressed:

M. Alivisatos (Athens) recorded 3,449 spinals without a death, he insisted upon the importance of not operating before the blood pressure had returned to nearly normal.

M. Danis (Brussels) considered the cause of headache to

be a continuation of leakage of C.S.F. through the wound in the dura caused by too large a needle.

M. J. L. Faure (Paris) pleaded warmly in favour of general anæsthesia by inhalation which he saw no reason to abandon.

M. de Rouville (Montpellier) after 28 years experience advocated spinal provided its possibilities were not exceeded, and considered it especially useful in gynæcological work.

M. Rocher (Bordeaux) advocated the use of this method in children between the ages of 5 and 15, but reported a case in which it was followed by hemiplegia in a boy of 13 years.

The following figures show the number of fatalities reported by various speakers:—

Total of cases.	Number of deaths.	Total of cases.	Number of deaths.
2000	3	3000	0
6147	3	82	1
2854	0	1213	1
750	11	2119	1
2000	0	331	2
6843	3	400	1
1000	0	346	3

A. L. Flemming.

ANÆSTHESIANA.

What They Think of Us, and What We Think of Ourselves.

In this department it will be our endeavour to include various expressions of opinions pertinent to the specialty of anæsthesia. If there are any bouquets among them they shall be duly presented. If, on the contrary, someone finds it advisable to hurl at us a brick-bat occasionally,it may do us good. So here goes.

Anæsthesia as a Specialty.

Of the various specialties one of the most select is that of the anæsthetist, and I have personal knowledge of some who, possibly inspired by their environment and provided with time for meditation while their surgeons are busy, have cultivated verse.

Sir Humphry Rolleston.

* * * *

The Introduction of Ether.

The introduction of ether into surgical operations was done by my hands. Mr. W. T. G. Morton, a dentist, of Boston called on me to say he had found the means of preventing pain in surgical operations, and he was so sanguine in regard to his new application, that I agreed to employ it on the first opportunity.

It was not until some time after I had used ether, that, in a conversation between Dr. Charles T. Jackson, Dr. Gould and myself, I learned that it was on the suggestion of Dr. Jackson that Mr. Morton was first led to use ether to prevent pain. A violent controversy subsequently took place between Jackson and Morton, and I was frequently appealed to for evidence on the subject. The amount of what I know may be comprised in a few words. Jackson suggested the use of ether to Morton, and Morton first employed it to prevent pain in the extraction of teeth; and, at his resquest, I first used it in a surgical operation. Dr. Jackson has also stated to me, that he advised Mr. Morton to apply to me to use it in a surgical operation.

"The Life of John Collins Warren, M.D." by Edward Warren, M.D., Boston, 1860.

No class of men needs friction so much as physicians; no class gets less. The daily round of a busy practitioner tends to develop an egoism of a more intense kind, to which there is no antidote. The few setbacks are forgotten, the mistakes are often buried, and ten years of successful work tend to make a man touchy, dogmatic, intolerant of correction, and abominably self-centred To this mental attitude the medical society is the best corrective, and a man misses a good part of his education who does not get knocked about a bit by his colleagues in discussion and criticisms. . . .

Osler.

* * * *

"Pat, what in the world is the matter?"
"I just got out of the hospital—was operated on for appendicitis."
"What's that got to do with the lump on yer head?"
"Why, it's got a lot to do with it. They run out of ether."

Life.

* * * *

Etherization

Æons on æons ago—
  (*"Breathe deep . . . breathe slow."*)
—I heard that wonderful chime,
  Exquisite—rhythmical—sweet!
Now t'is the knell of despair!
  God! How my mad pulses beat!
Fiends! Give me air—Give me air!
  Ha! Now, I'm climbing the sky!
Glorious! . . . the wind rushing by!
  (*"Breathe slow, Breathe deep."*)
Endless . . . that d-r-o-n-e of the plane,
  Ceaselessly maddening my brain!
Torture! . . . that aeroplane's song!
  Torture . . . for centuries long!
    Oh! . . . if I only could sleep!
Sleep? . . . (Suffocation, not sleep!)
  (*"Breathe slow, Breathe deep."*)
Down, . . . sinking down . . . ever down,
  Crushed by the weight of the world.
Honour and fame and renown,
  All, into nothingness hurled:
Self has no entity there;
  There is no future, no past,
Hatred, nor love, nor despair.
    (*"He is under—he's under at last!"*)

Operation

Sleep, all enfolding sleep!
Wrapped in oblivion deep,
The shuddering Spirit swoons its way.
Down the dark abyss, from the blessed Day
To the realms of the living dead,
Two worlds between;
While, in the skilled hand overhead
The saving knife cuts clean.
Eerie, and ghast, and lone
Is that gloomy borderland;
Unplumbed its depths; unseen, unknown,
Its drear and dismal strand.
Æons and æons elapse.
Light cannot penetrate here;
Blackness and darkness profound,
From the upper world no sound;
With the last link that snaps
Comes silence, that smites the ear. . . .

Ether Dreams

Re-incarnation

Deep, deep, fathoms deep,
Drowned men sleep their last, long sleep;
While, above them, evermore,
Rooted fast in ocean's floor,
Emerald fans of sea-weed grow,
Waving softly, to and fro.
Death! Is this Death? Am I *dead?*
Once, in the world overhead,
(Far above . . . O . . . far . . .
Ages and ages ago . . .
Something existed, called . . . "Me" . . .
—Now, there is naught but this sea,
Naught but these surges that roll,
Whelming me, body and soul,
Caught in the ebb and the flow—
Moving, supine, to and fro.
To . . . and fro . . . To . . and fro . . .
(*Breathe deep . . . breath slow . . .*'
Far above . . . O, . . . far . . .
Somewhat . . . called "Love"
Shines, . . . like a star.
If——Love——should—call——
I must leave . . all . . .
. . And . . .
Go!

Restoration

Through dark
A spark:
Through night
A light:
Far off
A call! . . .
Dear Love! I come, I come!
T'is past—all past!
Thank God!—at last
T'is Love,—and Life—and Home!

Edith J. Archibald, Halifax, N.S.
Can. Med. Assoc. Journ.

THE ANÆSTHETIST'S HOLIDAY.

As the days of the holiday season draw nearer many of us scan the horizon of possibilities in the way of going far afield on a limited purse. For the anæsthetist who can spare four or five weeks, the prospect of a trip to America and a visit to the great gathering of the International Anæsthesia Research Society and Associated Anæsthetists of the United States and Canada, which will be held at Chicago on October 14–18th, is sufficiently intriguing to excite one's fancy.

The recent innovation of comfortable accommodation at a reasonable charge brings a journey of this sort within the range of a modest purse; and the advantages of new contacts and visits to hospitals in the great cities of the American Continent are well worth considering when working out how best to spend the time and funds available for recreation purposes.

To those interested we commend the advertisement in this number of the White Star Line, while at the same time we would draw attention to the fact that where a sufficient number of delegates are going to the Congress there is a likelihood that a special reduction of 20 per cent off the regular fares may be allowed.

Tourist Class round trip rate is as low as £40 to which should be added the U.S. Head tax of £1 15s. 0d., and return rail fare from New York to Chicago £12 15s. 0d. Cabin and First Class rates are in proportion.